ABSTRACT

Elastin, the main component of arterial extracellular matrix, was thought to have a purely structural role. Consistent with this view, elastin hemizygous mice maintain arterial extensibility by increasing the number of elastic lamellae during development. However, mice lacking elastin die of obstructive arterial pathology. This pathology results from subendothelial proliferation and reorganization of smooth muscle, cellular changes similar to those observed in atherosclerosis. Thus, elastin is a molecular determinant of arterial morphogenesis and likely plays a central role in vascular disease. Mice which are heterozygous and null for the elastin gene have been developed. These mice are extremely useful for screening for drugs useful for treating persons with atherosclerosis, hypertension, SVAS or other vascular diseases.